CLINICO PATHOLOGICAL STUDY OF ASSOCIATED LESIONS IN BENIGN PROSTATIC HYPERPLASIA AND PROSTATIC ADENOCARCINOMA IN SURGICAL BIOPSY SPECIMENS

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CERTIFICATE

This is to certify that this dissertation entitled **"A CLINICO PATHOLOGICAL STUDY OF ASSOCIATED LESIONS IN BENIGN PROSTATIC HYPERPLASIA AND PROSTATIC ADENOCARCINOMA IN SURGICAL BIOPSY SPECIMENS"** is a bonafide work done by Dr.S.MANONMANI, in partial fulfillment of regulations of the **TAMIL NADU Dr.M.G.R. MEDICAL UNIVERSITY, Chennai**.

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DECLARATION

I declare that this dissertation entitled **"A CLINICO PATHOLOGICAL STUDY OF ASSOCIATED LESIONS IN BENIGN PROSTATIC HYPERPLASIA AND PROSTATIC ADENOCARCINOMA IN SURGICAL BIOPSY SPECIMENS"** has been conducted by me under the guidance and supervision of **Prof.Dr.P.Karkuzhali, M.D.,** in the Institute of Pathology and Electron Microscopy, Madras Medical College. It is submitted in partial fulfillment of the requirements for the award of the M.D. Pathology, September 2006 examination to be held under Dr.M.G.R.Medical University, Chennai. This has not been submitted by me for the award of any degree or diploma from any other University.

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CONTENTS

ABBREVIATION

AAH	-	Atypical adenomatous hyperplasia
ASAP	-	Atypical small acinar proliferation
BCH	-	Basal cell hyperplasia
BPH	-	Benign prostatic hyperplasia
CCCH	-	Clear cell cribriform hyperplasia
Cz	-	Central zone
H&E	-	Haematoxylin & Eosin
HPE	-	Histopathological examination
OP	-	Open prostatectomy
PAP	-	Prostatic acid phosphatase
PAS	-	Periodic acid schiff's
PCa	-	Prostatic carcinoma
PIN	-	Prostatic intra epithelial neoplasia
PSA	-	Prostatic specific antigen
PZ	-	Peripheral zone
TRUS	-	Trans rectal ultra sound
TURP	-	Transurethral resection of prostate
TZ	-	Transitional zone

INTRODUCTION

Benign prostatic hyperplasia and prostatic adenocarcinoma are the two principle conditions involving prostate among elderly men in urology practice. It accounts for more than 90% of all prostatic diseases. The prostate undergoes significant growth during fetal development, puberty and in most men during late middle age. At the end of puberty the prostate reaches approximately 26 g.

Benign prostatic hyperplasia occurs in individuals with intact testis and it is an androgen dependent disorder. The clinical incidence of this disease is only 8% during fourth decade, but it reaches 50% in the fifth decade and 75% in the eighth decade. BPH remains one of the major cause of obstructive urologic symptoms of patients coming to the urology out patient department. Transurethral resection of prostate remains the gold standard of treatment against which all other treatments of benign prostatic hyperplasia are measured. It is one of the most common procedure performed by urologists. After the introduction of screening for prostatic specific antigen, transrectal ultrasound and MRI prostate trucut needle biopsy became one of the most common urologic procedure to detect prostatic carcinoma.

In routine surgical pathology practice, making a morphological diagnosis of prostatic lesions, especially separating benign from malignant lesions is relatively straightforward. However, there are several glandular and stromal proliferations and normal histoanatomical structures which

may be mistaken for malignancy if one is not aware of the key histological features. It is very difficult to interpret these lesions in small tissue samples such as received in trucut needle biopsies.

For practicing pathologists and urologists there are currently two main issues in prostate pathology, one is the identification of prognostic factors that predict the outcome of individual patients with prostatic carcinoma. The other involves the early detection of prostatic carcinoma in the preinvasive phase. Hence, understanding the morphology of precursor or preinvasive lesions has become increasingly important.

The relationship between prostatic carcinoma and premalignant lesions of the prostate is a subject of great interest analyzed in numerous publications. Despite many studies conducted over this area each year, there were still important questions remain about the cause and prevention of prostate cancer.

Inspite of significant advances in the early detection of prostatic carcinoma by transrectal ultrasound, serum levels of prostatic specific antigen, the major diagnostic problem with the tumour pathology is making a diagnosis between adenocarcinoma and benign small acinar proliferations.

AIMS

- To find out the association of hyperplastic, metaplastic, inflammatory and premalignant lesions in transurethral resection of prostate, trucut needle and open prostatectomy specimens for a two year period from January 2004 to December 2005.

- To analyze the histological types of associated lesions in benign prostatic hyperplasia and prostatic adenocarcinoma.

- To evaluate the significance of these lesions in correlation with histological type and grading of prostatic adenocarcinoma.

REVIEW OF LITERATURE

Prostate is a pyramidal fibromuscular gland which surrounds the prostatic urethra from the bladder base to the membranous urethra and is itself surrounded by a thin but tough connective tissue capsule.

It lies at a low level in the lesser pelvis, behind the inferior border of the symphysis pubis and pubic arch, below the neck of the urinary bladder and anterior to the rectal ampulla through which it can be palpated[1].

Anatomically prostate is divided into five lobes anterior, posterior, median and two lateral lobes. The contemporary classification of the prostate into different zones was based on the work of Mc Neal. He divided prostate into peripheral zone (PZ), which lies mainly posteriorly and from which most carcinomas arise and a central zone (CZ), which lies posterior to the urethral lumen and above the ejaculatory ducts. There is also a periurethral and transitional zone (TZ) which is the most common location of benign prostatic hyperplasia[2].

The understanding of anatomical lobes and the surgical zones are essential in interpretation of prostatic biopsy specimens sent in Trans urethral resection of prostate (TURP) and trucut needle biopsy.

The prostate is supplied by branches from the inferior vesical, internal pudental and middle rectal arteries. It drains into vesical and internal illiac veins. Lymphatics from the prostate drain chiefly into internal

illiac, sacral and obturator nodes. The prostate has an abundant nerve supply from the inferior hypogastric plexus[1].

As stated in the introduction, benign prostatic hyperplasia occurs in men over the age of 50 yrs, and by the age of 60 yrs 50% men have histological evidence of benign prostatic hyperplasia.

Benign prostatic hypertrophy is a commonly used term. The term `nodular hyperplasia' proposed by Moore in 1936 in his classic study or the term `adenomyomatous hyperplasia', aptly describes the lesion.

BPH begins as micronodules in the transitional zone. They grow and coalesce to form macronodules around the inferior margin of the preprostatic urethra, just above the verumontanum. Macronodules in turn compress the surrounding normal tissue of the peripheral zone postero inferiorly thereby creating a `false capsule' around the hyperplastic tissue, which coincidentally provides a plane of cleavage for surgical enucleation of hyperplastic tissue by either transurethral resection of prostate or open prostatectomy.

BPH and adenocarcinoma arise in the men of the same age group. There are many associated epithelial and stromal lesions in benign prostatic hyperplasia which quite often mimic the histomorphology of adenocarcinoma. Hence, a depth of knowledge of these lesions is essential for correct histological diagnosis, clinical evaluation and followup.

Following is the working classification[3] of hyperplastic, metaplastic & tumour like lesions of prostate.

CLASSIFICATION

Epithelial lesions

Hyperplasia

- Benign nodular hyperplasia
- Basal cell hyperplasia
- Postatrophic hyperplasia
- Mesonephric remnant hyperplasia
- Clear cell cribriform hyperplasia
- Verumontanum mucosal gland hyperplasia

Metaplastic changes

- Squamous cell metaplasia
- Transitional cell metaplasia
- Mucinous metaplasia

Tumour like lesions

- Atrophy
- Sclerosing adenosis
- Nephrogenic adenoma of prostatic urethra
- Prostatic type urethral polyp
- Seminal vesicle/Ejaculatory duct/Cowper's gland/paraganglia in prostate samples

- Cysts
- Amyloidosis
- Calculi/calcification/cartilage

Stromal lesions

- Prostatitis
- Stromal hyperplasia/nodule
- Leiomyoma
- Fibroadenoma like lesion/phyllodes type tumor
- Post operative spindle cell nodule
- Inflammatory myofibroblastic tumor
- Blue nevus/prostatic epithelial pigment

Premalignant lesions

- Prostatic intraepithelial neoplasia
- Atypical adenomatous hyperplasia

EPITHELIAL AND STROMAL LESIONS

In 1936, Moore et al.[4] in his studies published a detailed account of the histological features of the normal and involutional prostate gland based on a study of 678 prostate glands. He suggested **atrophy** as a physiological and age related phenomenon. He described the presence of luminal acidophilic secretions in atrophy.

Post atrophic hyperplasia was first illustrated and published by Moore[4] in 1936 although he did not make any special reference to it in the text. This was followed by descriptions by Totten et al. in 1953, but they referred to this process as lobular hyperplasia. The term `post atrophic hyperplasia' and `post sclerotic hyperplasia' were coined by Frank et al. in 1954, who proposed this lesion as a precursor to prostatic adenocarcinoma. But later studies did not establish such a relationship between post atrophic hyperplasia and prostatic adenocarcinoma.

In 1999 Mahul B.Amin, Pheroze Tamboli, Muralivarma and John R.Srigley[5] studied 56 needle biopsy specimens containing 68 foci to ascertain the morphologic spectrum of postatrophic hyperplasia. Age of the patient ranged from 49 to 85 yrs. Selection of cases were restricted to those foci in which the process formed a small acinar proliferation atleast some of which raised the question of carcinoma. In this study, the prevalence of post atrophic atrophic hyperplasia presenting as a small acinar proliferation in consecutive biopsy specimens was 3.6%.

Basal cell hyperplasia was relatively a common lesion in hyperplastic prostates being examined by Young R.H. et al.[6] in his studies. **Clear cell cribriform hyperplasia** of the prostate is a rare form of hyperplasia found in BPH. It was recognized by the world health organization in 1980. Gleason[7] described the same lesions as a florid benign papillary cribriform hyperplasia in 1985. Immunoperoxidase studies demonstrated that the clear cells of CCCH show strong immuno reactivity for prostatic specific antigen and prostatic acid phosphatase.

Transitional cell metaplasia was studied by Yantiss R.K. Young, R.H.[8] in 1997 and incidence was found to be 34% in 103 consecutive prostatic biopsy specimens studied. They were quite commonly seen in the transitional zone around the prostatic urethra.

Mucinous metaplasia was studied by Frank et al.[9] in 1964. About 155 prostate glands were studied in which whole organ sections were stained for mucin. They described the presence of PAS and alcianblue positive goblet cells with in the transitional epithelium of prostatic urethra and proximal prostatic ducts. Dikman and Toker et al.[10] in 1973 noted the presence of seromucinous glands in prostatic stroma. They concluded that the lesion was an ectopia of minor salivary glands.

In 1993 David J. Grignon and Frances P.O Malley[11] observed tall columnar mucin secreting cells in 12 cases of benign prostatic hyperplasia out of 1700 cases in a three year study. All cases were stained histochemically for mucicarmine, alcianblue and period acid schiff's. They documented the presence of acid mucin in the luminae of basal cell hyperplasia, post atrophic hyperplasia and also in some glands involved with transitional cell metaplasia. The presence of acidic mucin in secretory cells in benign lesions point to the nonspecificity of this finding in the diagnosis of malignancy.

In 1999, Nickel JC, Downey J, Young I et al.[12] examined benign prostatic tissue sections and reported that inflammation was present in 44% to 100% of the prostatic tissue samples examined.

In 2003 Disilverio F, et al[14] analyzed retrospectively 3942 cases of benign prostatic hyperplasia for a period of 20 years and reported a mean patient age of 68.85± 7.67 years. Inflammation was found in 43.1% (1700 cases), predominantly it was a chronic inflammation. Incidence of focal acinar atrophy was significantly related to increase in age of the patient. They suggested that different histological features associated with benign prostatic hyperplasia were influenced by the age of the patients and volume of the prostate.

In 2005 Brian Difuccia et al.[15] tried to assess the variation of inflammation on single/multiple needle biopsy samples taken from predetermined areas of prostates removed from patients with cancer. They concluded that the foci of inflammation were predominantly multifocal and diffuse and occasionally periglandular in localization.

Sclerosing adenosis is a prostatic lesion which has been first reported by Chen A Schiff in 1983 as adenomatoid tumour of prostate due its morphological resemblance to adenomatoid tumour. The term sclerosing adenosis was introduced by Young & Clement in 1987 on the basis of histological resemblance to sclerosing adenosis of the breast. In 1991 Sakomoto et al.[16] analyzed 263 specimens of prostate, found 5 cases of sclerosing adenosis with an incidence of 1.9% which was found to be localized to the transitional zone.

PREMALIGNANT LESIONS

Two putative premalignant lesions of the prostate have been described. The clinical importance of recognizing these lesions is based on its strong association with carcinoma. Its identification in biopsy specimens of the prostate warrants further search for concurrent invasive carcinoma.

They are:

1. Prostatic intraepithelial neoplasia (PIN) (or)

   Ductal acinar/intra ductal dysplasia.

2. Atypical adenomatous hyperplasia (AAH) (or)

   Atypical small acinar proliferation (ASAP)

In 1965 Mc Neal[17] was the first one to describe this premalignant lesion, but his concept of preneoplasia was not fully embraced until 1980's. In 1986 Mc Neal & Bostwick[17], while studying 100 specimens of prostatic adenocarcinoma and 100 benign prostates obtained at autopsy, noticed foci of PIN in 82 prostates with carcinoma and 43 benign prostates. It provided strong support regarding the status of PIN as a precursor lesion.

In 1987 Brawer et al.[18] in his studies showed that there is a basal cell layer disruption in 56% of the cases with high grade PIN. The amount of disruption increased with increasing grades of PIN.

In 1989, there was a workshop on premalignant lesions of the prostate held at Bethesda Maryland and at this meeting **prostatic intra**

epithelial neoplasia was adopted as a preferred term. In 1989, Troncoso et al.[19] studied sections of prostate gland obtained from patients undergoing cystoprostatectomy for bladder cancer and found PIN in 89 of 100 prostates. Most of the PIN were multifocal and high grade and they were most common in prostates with carcinoma than without carcinoma.

From the practical point of view histological grading of PIN is important. Originally three grades were adopted. Then two grade system was established on the basis of cluster analysis. In 1989 Bethesda Workshop investigators agreed on the grading system that described low and high grade PIN by incorporating the previous grade 1 as low grade and grades 2 & 3 into the high grade[20].

In 1995 Montironi et al.[21] attempted to standardize the morphological criteria for identification and grading of PIN through a bayesian belief network. This network is designed to distinguish between normal prostate, low grade PIN, high grade PIN, cribriform adenocarcinoma and large acinar adenocarcinoma.

In 1996 David G.Bostwick M.D. et al.[22] in his clinical studies suggested PIN predates carcinoma by ten years or more and low grade PIN first appearing in men in their thirties. Finding of high grade PIN indicates the need for repeat biopsy and followup in particular, if prostatic specific antigen in serum is elevated.

In 1997 Junqi Qian, Peter Wollan, and David G.Bostwick[23], studied 195 whole mounted radical prostatectomy specimens with clinically

localized cancer. High grade PIN was identified in 86% of cases and was usually multicentric in 64.5% of cases and located in the non transitional zone in 63% and in all zones in 36% of cases.

In 1997 Nagle[24] RB et al. studied the progressive molecular changes in prostatic epithelium. Their data indicated that the dysplastic cells of PIN lesion and carcinomas were similar in nuclear and genomic features as well as protease expression.

In 2001, Saker WA, Partin[25] AW studied cases obtained at autopsy revealed that highgrade PIN was found in association with cancer in 63% to 94% of malignant and 25% to 43% of benign prostates. They suggested that there was a significant risk for patients with isolated highgrade PIN to have prostatic cancer which was confirmed by them on subsequent biopsies

In 2001 Allcaraz A, et al.[26] analyzed 28 high grade PIN cases found among 57 specimens of radical prostatectomy using a FISH technique to study the cytogenetic alteration and suggested that high grade PIN could be an early form of cancer.

In 2004, Rekhi B, Jaswal TS, Arora B.[27] studied PIN and AAH in ducto-acinar lining epithelium of 200 prostatectomy specimens. Out of 177 cases of nodular hyperplasia, 53 (29.9%) showed PIN and 38 (20.3%) showed presence of AAH. All 6 cases of pure carcinoma revealed foci of PIN (100%) suggesting a stronger association of high grade PIN as the potent precursor of carcinoma prostate.

In 1986, Mc Neal[28] referred **atypical adenomatous hyperplasia** (AAH) as a possible premalignant proliferation, most probably of carcinomas arising in the transitional zone. In 2000, Kien T.Mari et al.[29] reviewed 533 and 499 TURP specimens before and after the introduction of PSA screening respectively. They suggested the possibility of association of atypical adenomatous hyperplasia with low grade carcinoma arising from transitional zone and association of PIN with carcinomas arising in non-transitional zone.

Atypical adenomatous hyperplasia has been proposed as a precursor of prostatic adenocarcinoma of the transitional zone for the following reasons[20].

i. Age peak that precedes that of PCa.

ii. Increased incidence in association with PCa.

iii. Topographic relationship with small acinar PCa.

iv. Increased nuclear area and diameter.

v. A proliferative index similar to that of small acinar PCa.

vi. An occasional cases with genetic alterations.

The term `adenosis' was suggested to replace `atypical adenomatous hyperplasia'. This has not been accepted and `atypical' has been retained to indicate the unusual pattern of small acinar formations that characterize AAH. Currently, the term atypical adenomatous hyperplasia is replaced by `Atypical small acinar proliferation' (ASAP).

Previously the data were insufficient to conclude atypical adenomatous hyperplasia as a premalignant lesion. In 2005 Courtenay K.Moore et al.[30] in their study of 1,188 cases selected 105 cases of which 33 had HGPIN and 72 had ASAP. They applied a extended biopsy scheme over the patients diagnosed with high grade PIN and atypical small acinar proliferation. According to that study in the first repeat biopsy, only 1 of 22 (4.5%) men with previous HG PIN had cancer while 19 of 53 (36%) with a history of ASAP were found to have cancer. In the second repeat biopsy, none of the 11 men previously diagnosed with HG PIN had cancer. But 3 of 19 (16%) men with ASAP had cancer.

Hence, they suggested that highgrade PIN does not warrant a repeat biopsy and atypical small acinar proliferation continues to be associated with a high risk of cancer and requires atleast one repeat biopsy using extended biopsy scheme.

PROSTATIC ADENOCARCINOMA

Adenocarcinoma of prostate is the most common malignancy encountered in the urologic practice and is one of the leading cause of cancer death in men. Prostatic carcinoma is rare before 40 years of age, but the incidence rises quickly thereafter.

In 1817 Langstaff, et al.[31] were the earliest to recognize this entity; although at that time its differentiation from other enlargements of the prostate was imperfectly understood.

In 1900, Albarran and Halle et al. examined 100 cases and stated that adenocarcinoma prostate constituted 4% of all prostatic enlargements and defined adenocarcinoma as a lesion in between ordinary hypertrophy of old age and true prostate cancer.

Important questions always remain about the cause and prevention of prostate cancer, but many literature reviews show the significant advances in the understanding of premalignant epithelial lesions as well as clinching clinical techniques such as transrectral ultrasound and serum prostatic specific antigen levels enhance the early detection of prostatic cancer.

Histological grading

Historically, atleast 40 different classifications for prostatic carcinomas have been published, but only 19 have been examined closely for their prognostic significance. Broder et al.[32] is the pioneer who introduced the first grading system in 1925 in an attempt to estimate the malignant potential of cancer. It was based on degree of glandular differentiation, cell morphology, mitotic activity and degree of invasiveness.

In 1966 Gleason et al.[33] suggested a grading system which has been accepted by most of the urologists. He utilized glandular differentiation and growth pattern in relation to stroma to identify tumor grades.

M.D. Anderson et al.[3] grading system is based only on percentage of gland formation and tumour cell morphology is not taken into account.

In 1975 Mostofi et al.[34] proposed another grading system which was based on pattern of glandular differentiation and degree of nuclear anaplasia. It consists of three grades.

In 1980 Gaeta JF et al.[35] studied 19 cases of prostatic carcinoma and established a four grade system that combined the glandular pattern and nuclear morphology. This study showed a linear correlation between survival and mortality index.

In 1982 Brawn PN et al.[36] described a grading system based on percentage of tumour that shows differentiated (gland forming) and undifferentiated (non-gland forming) components. This grading system was also shown to correlate well with survival.

Galle et al.[37] compared the prognostic accuracy of five grading systems-Broders, Gleason, M.D.Anderson, Mostofi, Mostofi-schroeder in 50 prostatectomy specimens. They concluded that Gleason system had the lowest predictive ability. Whereas Broders and Mostofi-Schroeder systems had reasonable predictive ability.

Inspite of all these problems of intra and inter observer variability, imprecise predictive value, the **`Gleason Grading System'** remains the `de factor standard' for grading.

N.A. Epstein and L.P. FATTI[38], in 1976 evaluated morphological features in 146 cases of prostatic carcinoma which were correlated with 5

years survival, classifying it on the basis of Mc Neal's typing. They noted that if the tumour had indistinct cell borders and if there was no lymphocytic infiltration, then it was likely to have a very poor prognosis.

In 1976 Naomi A.Epstein[39], studied positive aspiration smears of 34 cases of histologically confirmed prostatic carcinoma. It was done to ascertain whether there was sufficient correlation between these features in aspiration smears and histological biopsies to enable prognosis to be assessed in cytological preparation.

He concluded that eventhough there was a 95% correlation in this study, the assessment of prognosis from morphological features was likely to be more accurate in sections than in smears, as the two features of prognostic significance-the presence or absence of cell borders and lymphocytic infiltration-can only be determined on sections.

In 1982, Alfred Bocking, et al[40]. formulated a new histological grading system for prostatic adenocarcinoma and tested its clinical significance. They concluded that survival probabilities of prostatic carcinoma patients were found to differ according to the actual least differentiated histological growth pattern. Survival time decreased with decreasing histological differentiation.

In 1986 Stacly E.Mills and Jackson E.Fowler[41], compared the Gleason histological score of prostatic carcinoma in biopsy specimens to corresponding radical prostatectomy specimens from 53 patients with

localized prostatic carcinoma. It was concluded that prostatic biopsy should be repeated when the initial diagnosis of adenocarcinoma is based on only limited quantities of neoplastic tissue with a low Gleason score and management decisions may be influenced by the true Gleason score.

There has been a little written about the diagnosis and reporting of adenocarcinoma of prostate diagnosed in core needle biopsy specimens. Jonathan I.Epstein[42] in 1996 evaluated the importance of reporting prostatic carcinoma in trucut needle biopsy specimens in correlation with the radical prostatectomy specimens over 499 cases. He noted that 66% (328 of 499) of the trucut needle biopsy specimens remained in the same grading group as the radical prostatectomy specimens and he explained the practical difficulty in reporting prostatic adenocarcinoma in trucut needle biopsies.

Anand P.Chokkalingam, et al.[43] in their study analyzed 86,626 men through a population based cohort study to assess the risk of prostatic carcinoma in 26 years, after the diagnosis of benign prostatic hyperplasia. They concluded that patients with BPH experienced excess risk of carcinoma (2% excess risk after 10 yrs. followup), when compared to general population. In contrast after first 5 years, patients with benign prostatic hyperplasia who did not receive surgical intervention had more risk compared to those who received treatment.

Prognostic factors of prostatic adenocarcinoma

1. Tumour related factors:

- Anatomic extent of disease
- Histologic grade

- Polidy
- Prostatic specific antigen (PSA)
- Prostatic acid phosphatase (PAP)

2. Patient related factors:

- Age
- Performance status-Pain, Obstructive symptoms
- Serum creatinine

3. Treatment related factors:

- Previous TURP
- Duration of primary response to hormone in progressive disease.

A number of studies have shown that prognostic factors for localized disease depends essentially on tumour related factors. Clinical staging and histological grading are amongst the most important and useful prognostic factors in prostatic cancer.

MATERIALS AND METHODS

An analysis of 520 cases of surgically resected prostatic specimens referred from urology department from January 2004 to December 2005 for a two year period, has been carried out in Institute of Pathology, Madras Medical College, Chennai.

The histological material were derived from biopsy specimens of transurethral resection of prostate in 447 cases, trucut needle biopsy in 62 cases and open prostatectomy in 11 cases. The age group of the patients ranged from 35-85 yrs.

Transurethral resection had been done to the patients who came to the urology out patient department with symptoms of urinary obstruction. If the patient had been clinically suspected of having carcinoma prostate by digital rectal examination, they were screened for prostatic specific antigen (PSA) level in the serum. Patients with elevated PSA levels in the serum or clinically suspicious patients or both have been selected for trucut needle biopsy. Only few patients underwent transrectal ultra sound (TRUS) guided biopsy.

As a routine, all prostatic specimens were fixed in 10% formalin. The total amount of prostatic chips received per each case varied grossly. But in general, we received 15 to 30 gm of prostatic chips for each case of TURP specimen. If the total amount of resected prostatic chips could be included in four histological sections it was examined in its entirety. Excess tissue was sampled at the rate of one histological section per 10 gram of resected tissue.

In most of the trucut biopsy specimens, we received only a bit of soft tissue measuring 0.5 to 1 cm and serial sections were taken from this. As a

routine, 5 to 8 histological sections were taken from open prostatectomy specimens.

All these histological sections were stained with **Haematoxylin & Eosin (H&E)** stain and examined. In each benign prostatic hyperplasia and adenocarcinoma case diagnosed, the evaluation was done on the following variables, i.e. associated inflammatory aspects, presence of focal acinar atrophy, metaplastic lesions, hyperplastic lesions and premalignant lesions.

All prostatic adenocarcinoma cases have been classified by the grading system of tumour differentiation described by Gleason et al. and thoroughly analysed for the presence of prostatic intra epithelial neoplasia (PIN) and atypical adenomatous hyperplasia (AAH).

Procedure of haematoxylin and eosin stain:

- Dewax the sections, hydrate through graded alcohols to water.
- Remove fixation pigments if necessary.
- Stain in haematoxylin for 5 minutes.
- Wash well in running tap water until sections become `blue' for 5 minutes.

- Differentiate in 1 percent acid alcohol for 2-4 sec.
- Wash well in running tap water until sections are again `blue' for 15 to 20 minutes.
- Stain in eosin for one minute.
- Wash in tap water for 5 minutes.
- Dry and mount the slide.

RESULTS AND ANALYSIS

The histological spectrum of all associated epithelial and stromal lesions in benign prostatic hyperplasia and adenocarcinoma have been analyzed using various statistical tests. Out of 520 cases analyzed, 447 were TUPR specimens, 62 were trucut needle biopsies and 11 were open prostatectomy specimens. In total about 4619 cases of BPH and 41 cases of adenocarcinoma have been diagnosed in this two year period.

Table: 1

Distribution of cases in relation to surgical biopsy specimens

Surgical biopsy specimens	Total No. of cases	Percent
Transurethral resection of prostate	447	86%
Trucut needle biopsy	62	12%
Open prostatectomy	11	2%

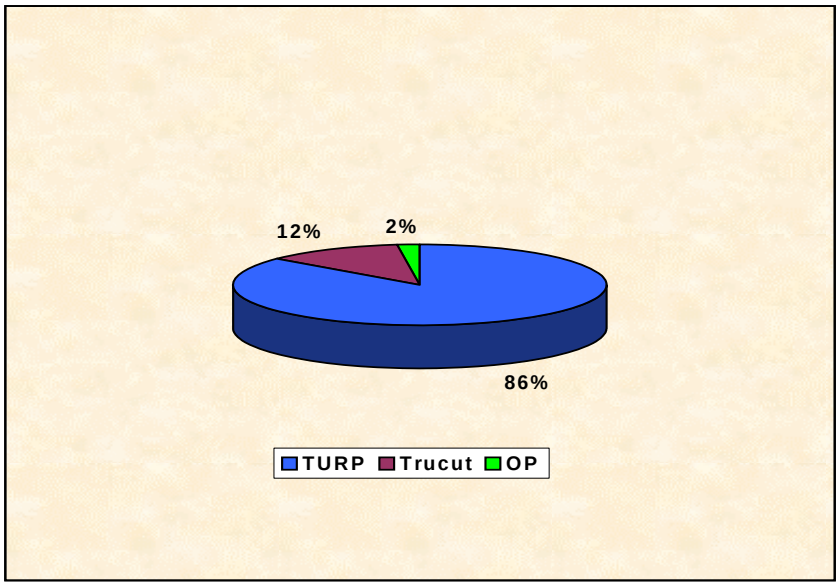


Table : 2

Distribution of cases in relation to Age

Age group	No. of cases	Percent
31-40 Yrs	6	1.1%
41-50 Yrs	31	5.9%
51-60 Yrs.	127	26.3%
61-70 Yrs.	214	41.5%
70-80 Yrs.	118	22.6%
81-90 Yrs	14	2.6%

Table : 3

Distribution of cases based on HPE diagnosis

Lesions	Total no. of cases	TURP	Trucut biopsy	Open prostatectomy
BPH	461 (88.6%)	429 (96%)	21 (33.8%)	11 (100%)
Adenocarcinoma	41 (7.8%)	18 (4%)	23 (37.2%)	0
Inadequate tissue	18 (3.6%)	0	18 (29.0%)	0

Table : 4

EPITHELIAL CHANGES IN BENIGN PROSTATIC HYPERPLASIA

S.No.	Lesion	No. of cases	Percent
1.	Focal acinar/cystic atrophy	35	7.5%
2.	Squamous cell metaplasia	13	2.8%
3.	Transitional cell metaplasia	23	5.0%
4.	Mucinous metaplasia	1	0.2%
5.	Post atrophic hyperplasia	2	0.4%
6.	Basal cell hyperplasia	26	5.6%
	Complete	14	3.0%
	Incomplete	12	2.6%
7.	Clear cell cribriform hyperplasia	2	0.4%

Table : 5

STROMAL CHANGES IN BENIGN PROSTATIC HYPERPLASIA

S.No.	Lesion	No. of cases	Percent
1.	Chronic Inflammation	132	28.6%
	Lymphocytic	125	27.1%
	Granulomatous	7	1.5%
2.	Stromal nodule/hyperplasia	32	7.0%
3.	Stromal calcification	1	0.2%
4.	Leiomyomatous nodule	3	0.6%
5.	Infarction	3	0.6%
6.	Abscess	4	0.8%

Putative premalignant lesions

Out of the putative premalignant lesions diagnosed in association with benign prostatic hyperplasia and prostatic adenocarcinoma, PIN changes constituted the major category. Among these, highgrade PIN was the most prevalent premalignant lesion diagnosed in cases of adenocarcinoma. Low grade PIN was more prevalent than high grade PIN in benign prostatic hyperplasia.

Atypical adenomatous hyperplasia was most commonly seen in cases diagnosed with benign prostatic hyperplasia. It was diagnosed only in one case of adenocarcinoma (see Table 6). Highgrade PIN changes were most commonly seen in trucut biopsy specimens in association with prostatic adenocarcinoma. Low grade PIN changes and atypical adenomatous hyperplasia were predominantly seen in TURP specimens (see table 7).

Table : 6

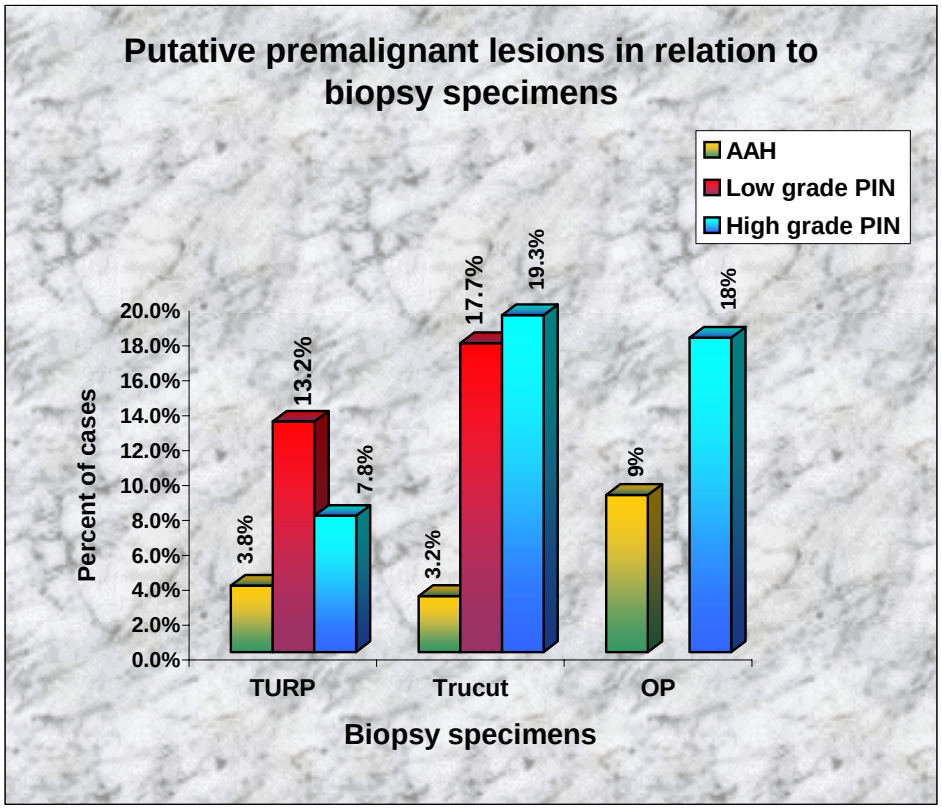
DISTRIBUTION OF PREMALIGNANT LESIONS IN RELATION TO HPE DIAGNOSIS

Lesion	AAH	PIN	Low grade PIN	High grade PIN
BPH	19 (4.1%)	90 (19.5%)	62 (13.4%)	28 (6.1%)
Adenocarcinoma	1 (2.4%)	29 (70.7%)	8 (19.5%)	21 (51.2%)

Table : 7

PUTATATIVE PREMALIGNANT LESIONS IN RELATION TO BIOPSY SPECIMENS

Lesions	No. of cases	TURP	Trucut biopsy	Open prostatectomy
AAH	20 (3.8%)	17 (3.8%)	2 (3.2%)	1 (9%)
PIN Changes	119 (23%)	94 (21%)	23 (37%)	2 (18%)
Low grade PIN	70 (13.5%)	59 (13.2%)	11 (17.7%)	0
High grade PIN	49 (9.5%)	35 (7.8%)	12 (19.3%)	2 (18%)



Prostatic adenocarcinoma

Total number of adenocarcinoma cases diagnosed for a period of two years 41 cases.

Table : 8

DISTRIBUTION OF MALIGNANT CASES IN RELATION TO BIOPSY SPECIMENS

Biopsy specimen	No. of cases	Percent
TURP	18	44%
Trucut	23	56%

Table :9

DISTRIBUTION OF ADENOCARCINOMA CASES BASED ON TUMOUR GRADING

Grade	No. of cases	Percent
I	10	24.3%
II	12	29.3%
III	14	34.2%
IV	4	9.8%
V	1	2.4%

DISCUSSION

Benign prostatic hyperplasia and prostatic carcinoma, the two common urologic conditions among elderly men, share not only a similar hormonal milieu within the prostate, but also several epidemiologic and clinical factors. Both conditions increase with advancing age, both require androgens for growth and development and both respond to antiandrogenic therapy. Both share similar putative risk factors such as insulin-like growth factors, insulin and obesity.

Most of the patients, came with complaints of symptoms of urinary obstruction to the urology O.P. such as difficulty in micturition, hesitancy, frequency, pain during micturition and feeling of incomplete voiding. Patients suspected of having prostatic carcinoma on digital rectal examination were screened for prostatic specific antigen and if found to have elevated levels of prostatic specific antigen underwent trucut needle biopsy. Few patients were biopsied under the guidance of Transrectal ultra sound (TRUS).

Out of 520 cases analyzed in our study benign prostatic hyperplasia were found in 461 (88.6%) patients, prostatic adenocarcinoma in 41 (7.8%) patients and 18 (3.6%) trucut biopsy specimens turned out to be inadequate samples for making a diagnosis (See table-3). The patients were in the age group ranging from 35 to 85 yrs. The peak age group was 61 to 70 yrs, followed by 51-60 yrs (See table-2).

Epithelial lesions

The total no. of 461 cases diagnosed with benign prostatic hyperplasia were analyzed for all other associated hyperplastic, metaplastic and atrophic lesions. Out of these lesions commonest were the focal acinar atrophy constituting 35 cases (7.5%) followed by basal cell hyperplasia in 26 cases (5.6%) and transitional cell metaplasia in 23 cases (5.0%). Basal cell hyperplasia were classified into complete and incomplete forms with both the types showing almost equal incidence. Mucinous metaplasia and clear cell cribriform hyperplasia constituted less than 1% of cases but were of histomorphological interest.

Di Silverio F. et al.[14] studied 3942 patients with benign prostatic hyperplasia and observed that focal acinar/cystic atrophy was found significantly increased according to the patient age in decades. In concurrence with the above study this study also showed 7.5% cases with cystic/acinar atrophy showing an increased incidence towards the higher age group.

Mahul B.Amin et al[5]. stated that the most commonly encountered pattern simulating the microacinar architecture of carcinoma is atrophy. A less common glandular pattern that forms part of the spectrum of atrophy is postatrophic hyperplasia, a lesion that has attained a renewed attention in the recent past in the literature.

Recently, several prostatic lesions composed of small glandular pattern mimicking adenocarcinoma have been identified or have had their diagnostic criteria refined. These include basal cell hyperplasia, sclerosing adenosis, atypical adenomatous hyperplasia (adenosis), post atrophic hyperplasia, hyperplasia of mesonephric duct remnants, verumontanum mucosal gland hyperplasia and nephrogenic adenoma of prostatic urethra secondarily involving prostatic parenchyma.

The small acinar pattern is commonly a source of consultation material, and approximately 5% to 10% of the cases in our study have had a preliminary diagnosis of carcinoma or a serious consideration of it.

So an awareness and firm understanding of the morphological spectrum of these epithelial lesions is of critical importance, especially in prostatic trucut needle biopsy specimens in which it shows some overlapping features with small acinar adenocarcinoma. And also the tissue sample obtained from the representative area is less and so the interpretation becomes difficult.

Many small acinar lesions of the prostate alluded to earlier have fairly consistent histological factors, and hence, there is little variation in their morphological expression. But post atrophic hyperplasia represents the process of hyperplasia and atrophy in transition and has less constant histology. The recognition of post atrophic hyperplasia in trucut needle biopsies is likely to be relatively straight forward, if the entire focus containing atrophic and hyperplastic glands is seen. Problems may arise when entire lesion is not represented.

Since basal cell hyperplasia has a distinctive microscopic appearance, its recognition is not difficult in most cases, but it may mimic adenocarcinoma of small acinar pattern, particularly if one is not familiar with this histological entity. BCH is characterized by a nodular growth of nests, tubules and cords filled with proliferating, small, darkly staining basal cells. Most of the nests of basal cell hyperplasia show vertical palisading of basal cells towards the periphery.

Young R.H. et al.[6] study on prostatic lesions proved that basal cell hyperplasia is relatively a common lesion in hyperplastic prostates examined. This study also showed 26 cases (5.6%) and is more in number when compared to other hyperplastic and metaplastic lesions.

Squamous metaplasia of the prostatic glands was made out in 13 (2.8%) cases and in three cases it was identified next to an area of infarct. Out of the 23 (5%) cases of transitional cell metaplasia most of the cases were found in the periurethral region/transitional zone, similar to the study done by Yantiss R.K. and Young, R.H[8].

Clearcell cribriform hyperplasia is a rare entity made out only in two cases and it is important that CCCH should not be mistaken for a carcinoma or preneoplastic condition of the prostate with a papillary-cribriform pattern. The key to the diagnosis of CCCH is the combination of bland cytology and architectural uniformity.

In all these cases with small glandular proliferation basal cell layer is present. In problematic cases high molecular weight cytokeratin (Clone 34β E12) can be applied which will help accentuate the basal cells and confirm the benign diagnosis.

Frank et al.[9] in their studies noted the alcianblue positivity in the luminal secretions of adenocarcinoma and they advocated this as a valuable aid to a make a diagnosis of well differentiated carcinoma.

Later studies done by David J.Grignon et al.[11] have shown alcianblue positivity in acid mucin secretions in the lumina of basal cell hyperplasia, post atrophic hyperplasia, atrophy, sclerosing adenosis and in transitional cell metaplasia. So the acid mucin positivity can not be taken as a specific entity in making a diagnosis of carcinoma.

Hence it is stated that once a small acinar proliferation is identified, it need not be included in the final pathological diagnosis. However, it is recommended a mention should be made of this finding in the microscopic features, particularly if carcinoma is considered in the clinical/pathological evaluation.

More over these are not a distinct clinicopathological entities, but merely a pattern in the morphological spectrum which mimic cancer. So appreciation of key histological features is highly essential to make a reliable separation of small acinar carcinoma from there associated lesions which will reduce the diagnosis of `atypia' and `suspicious of carcinoma' in

transurethral resection of prostate and especially in trucut needle biopsy specimens.

Stromal lesions

All the cases diagnosed with benign prostatic hyperplasia were analyzed for stromal lesions. Proliferative stromal lesions were noticed in 32 cases (7.0%). Chronic inflammation was the commonest lesion noticed in 132 cases (28.6%) out of which lymphocytic inflammatory infiltrate was seen in 125 cases ((27.1%) followed by granulomatous inflammation in 7 cases only (1.5%). Stromal calcification was noticed in only one case (0.2%). Infarction and abscess constituted less than 1% of cases.

Di Silverio F. et al.[14] study on 3942 patients with histopathological diagnosis of benign prostatic hyperplasia, the mean patient age was 68.85 ± 7.67 yrs. In particular, inflammatory changes were associated with BPH in a high percentage of the cases (43.1%=1700 cases). In our study about 132 cases having chronic inflammation were associated with BPH.

Out of 132 cases which showed inflammatory infiltrate the aspect of involvement with inflammatory reaction varied significantly according to prostate volume especially in case of lymphocytic infiltration. There was a trend to increase with increase in prostate volume. The inflammatory cells present in the tissue were primarily lymphocytes, monocytes, activated macrophages and mast cells.

Brian Difuccia et al.[15] in his study demonstrated that the distribution of inflammation was more variable with multifocal and diffuse patterns being the most common and periglandular being less common. Multifocal inflammation was present in 48 to 68% of cases and diffuse inflammation was present in 24% of cases. Focal inflammation was only present in 4 to 16% of cases.

Similar to the above study, evaluation of our cases showed 60 to 80% with multifocal involvement of the tissue sample and 20 to 40% showed diffuse infiltration by chronic inflammatory cells. The severity of the inflammation also varied a lot.

A large variation in distribution of inflammation was found between microscopic fields of the same biopsy and also between different areas of the same blocks of prostatic tissue. One interesting case of tuberculous granulomatous prostatitis was also diagnosed in this study.

The evaluation of prostatic pathological findings with prostatitis has been problematic since they are only incidental findings in biopsies, which were performed for various other reasons. The diagnosis and treatment of prostatitis do not warrant surgical removal or biopsy of the prostate. Biopsy of these patients are rarely indicated or performed.

Stromal hyperplasia is most often seen in the setting of benign prostatic hyperplasia. It is characterized by bland spindle cell proliferation

devoid of glandular elements. The distinction between stromal nodule and a leiomyoma is some what arbitrary.

Prostatic leiomyoma has not been universally accepted as a distinctive entity. Less than 70 cases of solitary leiomyoma has been reported in medical literature[3]. In this study three leiomyomatous nodules were diagnosed. Many other stromal lesions like phyllodes type tumour, blue nevus and fibroadenoma like lesion were not diagnosed in this study.

Premalignant lesions

Since the introduction of prostate specific antigen (PSA), trucut needle biopsy has become one of the most common urological procedures. More than that, transrectal ultrasound directed biopsy permits the localization of the needle and the tissue being sampled. Often the only diagnosis rendered is that of highgrade prostate intra epithelial neoplasia (HGPIN) or atypical adenomatous hyperplasia (AAH)/atypical small acinar proliferation (ASAP).

PIN and AAH are considered as a putative premalignant lesions of adenocarcinoma. Prostatic intraepithelial neoplasia is defined as architecturally benign ducts and acini lined by abnormal secretory cells with changes similar to those in cancer. Atypical adenomatous hyperplasia denotes the presence of suspicious glands with insufficient cytological or architectural atypia for a definitive cancer diagnosis.

The morphological continuum of PIN that results in early invasive adenocarcinoma is now divided into two grades, low grade and high grade replacing the previous three grade system. PIN 1 is considered as low grade and PIN 2 and 3 are considered as high grade.

Diagnostic criteria of PIN

	Features	Lowgrade PIN	High Grade PIN
A.	Architectural features	Epithelial cell crowding with irregular spacing	Similar to low grade, more crowding & stratification
B.	Cytological features		
	• Nuclei	Enlarged with considerable size variation	Enlarged with some size and shape variation.
	• Chromatin	Normal	Increase in density and clumping
	• Nucleoli	Rarely prominent	Large and prominent, sometimes multiple
C.	Associated features		
	• Basal cell layer	Intact	May show disruption
	• Basement membrane	Intact	Intact

McNeal & Bostwick[17] study of 100 specimens of prostatic adenocarcinoma and 100 benign prostates obtained at autopsy, identified PIN in 82 prostates with carcinoma and 43 prostates without carcinoma.

Troncoso et al.[19] studied sections from 100 prostate glands obtained from patients undergoing cystoprostatectomy for bladder carcinoma. He found PIN in 89 cases out of 100 prostates. Most of the PIN were multifocal and high grade.

Similar to the above studies, this study showed low grade PIN in 62 cases (13.4%) of benign prostatic hyperplasia and in 8 cases (19.5%) of adenocarcinoma. High grade PIN were identified in 28 cases (6.1%) of benign prostatic hyperplasia and 21 (51.2%) cases of adenocarcinoma. This study also showed a high incidence of high grade PIN in cases of prostatic adenocarcinoma.

This fact has been emphasized by Junqi Qian, M.D. et al.[23] study of 195 radical prostatectomy specimens with clinically localized cancer. They identified PIN in 86% of prostates and it showed increased incidence of PIN with prostatic adenocarcinomas.

An overview of these studies show the clinical importance of recognizing PIN is actually based on its strong association with prostatic carcinoma. Prostatic intraepithelial neoplasia coexists with carcinoma in most cases. It retains an intact or fragmented basal cell layer unlike carcinoma which lacks basal cell layer.

Montironi & Schulman et al.[20] have suggested that a small subset of basal cell layer houses the stem cell population. These cells are the presumptive origin of prostatic intraepithelial neoplasia and prostatic adenocarcinoma.

The histopathological continuum that culminates in high grade PIN and early invasive carcinoma is characterized by progressive basal cell layer disruption, changes in expression of markers of secretory differentiation, nuclear and nucleolar abnormalities, increasing microvessel density, cell proliferation, DNA content and allelic loss.

Virtually all phenotypic and genotypic studies of high grade PIN have indicated that it is more closely related to carcinoma than to benign epithelium. High grade PIN on its own has little or no significant influence on serum prostatic specific antigen concentration and does not cause clinically suspicious elevations.

Rekhi B. et al.[27] studied 200 prostate specimens out of which 177 cases of BPH, showed PIN changes in 53 (29.9%) cases and AAH in 38 (20.3%) cases. All 6 cases (100%) of pure carcinoma revealed foci of PIN.

In this study out of 461 cases of benign prostatic hyperplasia, prostatic intraepithelial neoplastic (PIN) changes were identified in 90 cases (19.5%) and atypical adenomatous hyperplasia were identified in 19 cases (4.1%). Out of 41 cases of adenocarcinoma of prostate a total of 29 cases (70.7%) showed PIN changes.

Atypical adenomatous hyperplasia is a small acinar proliferation that is usually seen in intimate association with nodular hyperplasia in the transitional zone, often appearing at the periphery of a nodule. It shares many of the cytological features of the adjacent larger benign acini and is

separated from well differentiated carcinoma by the presence of inconspicuous nucleoli infrequent crystalloid and a fragmented basal cell layer.

Nucleolar size is invariably smaller in atypical adenomatous hyperplasia than adenocarcinoma. Despite the utility of these features, the absolute distinction between AAH and carcinoma is difficult in some cases. This may possibly be a transition between normal prostate epithelium and well differentiated adenocarcinoma usually seen in the transitional zone.

Kien T. Mai[29] in his study of 533 and 449 prostate specimens studied before and after the introduction of PSA screening respectively suggested that atypical adenomatous hyperplasia may be related to a subset of carcinoma that arises in transitional zone in association with benign prostatic hyperplasia.

In this study only one case (2.4%) of adenocarcinoma diagnosed in trucut needle biopsy showed the presence of atypical adenomatous hyperplasia. Other 19 cases (4.1%) of AAH were found in benign prostatic hyperplasia showing the representation from transitional zone.

There are many histological mimics for PIN such as atypical basal cell hyperplasia, cribriform hyperplasia and metaplastic changes associated with radiation and infarction. Atypical adenomatous hyperplasia can be confused with simple lobular atrophy, post atrophic hyperplasia, sclerosing

atrophy, basal cell hyperplasia and verumontanum mucosal gland hyperplasia.

From this study it has been shown that the frequency of PIN in prostates with cancer is significantly increased when compared with benign prostates. High grade PIN was identified in 21 (51.2%) cases of adenocarcinoma and 28 cases (6.1%) of benign prostatic hyperplasia. It corresponds with many studies conducted previously per literature.

The data linking atypical adenomatous hyperplasia and carcinoma are limited and inconclusive. If AAH is a precursor to a subset of prostatic carcinoma, that subset of prostate carcinoma is most likely to be a low grade carcinoma in the transitional zone. High grade PIN provides the highest risk ratio of all known predictive factors and the identification of PIN in biopsies predicts the presence of carcinoma in subsequent biopsies. The risk of adenocarcinoma in subsequent biopsies is 15 times greater in patients with high grade PIN.

This finding indicates the need for repeat biopsy and follow up when high grade PIN is identified in a biopsy, especially in patients with an elevated serum prostatic specific antigen concentration. Studies to date have not determined whether PIN remains stable, regresses or progresses, though there is an implication that it can progress.

Prostatic adenocarcinoma

Prostatic adenocarcinoma is often a multicentric malignant process consisting of two rather distinct types of PCa with different origins:

1. Non transitional zone prostatic carcinoma (Non-TZPCa)
2. Transitional zone prostatic carcinoma (TZPCa)

Non transitional zone prostatic carcinoma is a tumour that is accessible to digital rectal examination and trans rectal ultrasound and is associated with high grade carcinoma, PIN and a high incidence of tumoral invasion into the prostatic capsule, perineural spaces and seminal vesicles.

In contrast transitional zone prostatic carcinoma is characterized by a low grade acinar pattern of carcinoma that is rarely associated with capsular invasion and seminal vesicle involvement. It is most likely to arise from prostatic epithelium often in association with atypical adenomatous hyperplasia (AAH). Furthermore, AAH is more commonly identified in the transitional zone where as PIN is more prevalent in peripheral zone.

Due to the respective locations of these two types of carcinoma, a large proportion of incidental adenocarcinomas identified in transurethral resection of prostate is of TZ carcinoma type. With the introduction of prostatic specific antigen as a screening test for prostatic carcinoma, the incidence of PCa has been increased. This phenomenon was due to the early detection of prostatic carcinoma from the existing pool of carcinoma and not due to a net increase in the number of new cases.

Histological classification of tumours discriminate neoplasms according to their different histogenetic derivations primarily and allow predictions about the biological behaviour of the tumors. Histologic grading systems subdivide tumours of the same histogenetic origin into groups with different degrees of malignancy and different prognosis.

The prognostic significance of a grading system should be accepted only if a good correlation of tumour grades with survival probabilities of the different groups of patients can be demonstrated. Thus the clinical relevance of a grading system is established by correlating individual tumour grades with the clinical course of the disease.

Basic conditions for achieving this goal are:

1. that each applied diagnostic criterion should correlate with tumour malignancy and prognosis.

2. that the subjective grading results should be sufficiently reproducible

3. that the grading results obtained from biopsies should be sufficiently representative of tumour as a whole.

Out of 40 different histological classifications published, 19 have been examined closely for the prognostic significance. The Gleason's histological grading system has been accepted world wide as a grading system for prostatic carcinoma.

GLASON'S GRADING SYSTEM FOR PROSTATIC ADENOCARCINOMA

Score	Appearance of glands	Size of glands	Architecture of glands	Peripheral borders	Stromal invasion	Cytoplasm
1	Simple, round, monotonously replicated	Medium, regular	Closely packed, rounded masses	Circumscribed, pushing and expansile	Minimal	Similar to benign epithelium
2.	Simple, round, some variability in shape	Medium, less regular	Loosely packed, rounded masses	Less circumscribed, early infiltration	Mild, with definite separation of glands by stroma	Similar to benign epithelium
3.	Angular, with variation in shape papillary and cribriform	Small to Medium, Irregular	Variably packed	Ragged infiltration	Marked	More basophilic than (1) and (2)
4.	Micro acinar papillary & cribriform	Irregular	Fused in Chains & Cords	Ragged infiltration	Marked	Dark, may be clear (hypernephroid)
5.	Few (or) no glands comedonecrosis may be present		Fused in sheets and masses	Ragged infiltration	Marked	Variable

Based on the above features, the predominant primary pattern and the secondary pattern seen are scored and are summed up to produce the total score. Hence, the possibilities of total scores range from 2 to 10.

- Low grade carcinomas

  Gleason's score of 6 or less than that

- High grade carcinomas

  Gleason's score of 7 more than that

Gleason's score in variants of prostatic adenocarcinoma

The variants of prostatic carcinoma account for fewer than 10% of cases and rarely occur in pure form.

The variants are:

- Ductal endometrioid carcinoma without necrosis-(Gleason score 3) with necrosis (Gleason score 5)
- Small cell undifferentiated carcinoma (Gleason score 5).
- Mucinous carcinoma (Gleason score 4)
- Signet-ring cell carcinoma (Gleason score 5)
- Sarcomatoid carcinoma (Gleason score 5)
- Carcino sarcoma (Gleason pattern 5)
- Adenoid basal cell tumour (Gleason score 3 to 5).
- Lymphoepithelioma like carcinoma (Gleason score 5).
- Adenocarcinoma after androgen deprivation therapy (Gleason score 4 & 5).

Over the years, the grading system has stood the test of time and numerous studies have shown it to correlate strongly with survival rates, lymphnode metastasis, tumour volume, clinical stage and pathological stage.

Moreover Gleason's histological grading system is simple, easier to use and is confirmed by Stacey E.Millis, et al.[41] and Jonathan I.Epstein, et al.[42] and by many investigators as satisfactorily reproducible.

In this study, Gleason's grading system is applied over 41 cases of prostatic adenocarcinoma, diagnosed in these two year period of 2004 and 2005. Out of which Grade III tumurs were the commonest noticed in 14 cases (34.2%) followed by Grade II in 12 cases (29.3%) and Grade I in 10 cases (24.3%).

In this study out of 41 cases of adenocarcinoma reported more number (23 cases 56%) of malignant cases have been reported from trucut needle biopsy. Out of 62 trucut needle biopsies studied, 23 cases (37.2%) were diagnosed with adenocarcinoma, 21 cases (33.8%) with benign prostatic hyperplasia and 18 (29%) biopsy samples turned out to be inadequate or unsatisfactory. The last group of samples contained only few glands, fibrous tissue and/or blood clot only.

The diagnostic accuracy of prostatic adenocarcinoma in trucut needle biopsy is hampered by the less quantity of material available and numerous benign lesions that mimic adenocarcinoma histologically.

At times the limited biopsy material may lead to underestimation of Gleason's score and there by depriving the patients from getting the correct treatment.

In prostatic biopsy specimens, pathologists are not only called upon to diagnose carcinoma but also to quantify and grade these cancers accurately. This is to provide better prognostic information to the patients and also to inform the clinicians as to whether the tumour can be followed expectfully, treated definitively or to be treated palliatively because of its advanced clinical stage.

SUMMARY AND CONCLUSION

- Among all the surgical specimens received for the study period of two years in the Institute of pathology, the most common were TURP specimens with benign prostatic hyperplasia (92.1%) constituting the commonest histological category. Adenoarcinomas were found in 7.9% of cases. Most of the adenocarcinomas were diagnosed in trucut needle biopsy (56%).

- About more than 90% of prostatic lesions studied were found in sixth to eighth decade.

- All cases of focal acinar/cystic atrophy showed increase in trend towards increasing age in decades.

- Among the hyperplastic lesions, basal cell hyperplasia was found to be the most common epithelial lesion (26 cases) compared to the other lesions such as postatrophic hyperplasia, clear cell cribriform hyperplasia etc.

- Transitional cell metaplasia was found to be the most common metaplastic lesion (23 cases) and about half of the cases were found in association with chronic prostatitis. About one fourth of the cases of squamous cell metaplasia were found in the periphery of areas of an infarct.

- Chronic inflammation was the most common stromal lesion

noticed in 145 cases (27.8%) predominantly it is a lymphocytic inflammatory infiltrate with multifocal and diffuse distribution seen in association with benign prostatic hyperplasia in 28.6% cases and with adenocarcinoma in 31.7% cases. One interesting case of tuberculous granulomatous prostatitis was also diagnosed.

- Among the premalignant lesions, PIN changes (70.7%) were found most commonly in association with prostatic adenocarcinoma. Out of which high grade PIN (51.2%) was the commonest premalignant lesion observed.

- Though atypical adenomatous hyperplasia is considered to be a putative premalignant lesion, contrarily it was observed only in one case (2.4%) of prostatic adenocarcinoma. Most commonly it was observed in 19 cases (4.1%) of benign prostatic hyperplasia.

- In concurrence with the literature it was observed that high grade PIN was the most common premalignant lesions associated with prostatic adenocarcinoma (51.2%) rather than atypical adenomatous hyperplasia. AAH was mostly observed in transitional zone and is probably a precursor of low grade adenocarcinomas arising in transitional zone.

MASTER CHART

ɔ.	Biopsy No.	Age/Male	Biopsy Specimen	Histopathological Diagnosis	Associated Lesions
	85/04	70	TURP	BPH	
	86/04	58	TURP	BPH	Cystic atrophy, chronic prostatitis

ɔ.	Biopsy No.	Age/Male	Biopsy Specimen	Histopathological Diagnosis	Associated Lesions
	87/04	39	TURP	BPH	Chronic prostatitis, squamous metaplasia, transitional cel
	152/04	51	TURP	BPH	Stromal nodule
	160/04	55	TURP	BPH	Chronic prostatitis
	162/04	62	TURP	BPH	Cystic Atrophy
	202/04	75	TURP	BPH	Cystic Atrophy
	291/04	70	TURP	BPH	
	293/04	67	TURP	BPH	Squamous metaplasia, mucinous metaplasia
	345/04	65	TURP	BPH	Transitional cell metaplasia
	346/04	65	TURP	BPH	Chronic prostatitis
	351/04	50	TURP	BPH	Chronic prostatitis
	609/04	69	Trucut	BPH	
	621/04	60	TURP	BPH	
	692/04	55	TURP	Adenocarcinoma Grade III	High Grade PIN
	716/04	60	TURP	BPH	Cystic atrophy, transitional cell metaplasia
	717/04	73	TURP	BPH	High grade PIN
	719/04	70	Trucut	Inadequate tissue	
	736/04	80	TURP	BPH	
	821/04	60	Trucut	Adenocarcinoma Grade II	Low grade PIN
	822/04	55	Trucut	BPH	
	831/04	61	TURP	BPH	Cystic atrophy
	881/04	74	TURP	BPH	Chronic prostatitis
	882/04	75	TURP	Adenocarcinoma Grade III	High grade PIN
	936/04	70	TURP	BPH	Chronic prostatitiss
	951/04	60	Trucut	Adenocarcinoma Grade IV	High grade PIN
	973/04	55	TURP	BPH	Stromal nodule
	974/04	50	TURP	BPH	Chronic prostatitis
	996/04	65	TURP	BPH	
	1092/04	60	Trucut	Tiny Tissue	
	1097/04	70	Trucut	Only fibrous tissue	
	1105/04	62	TURP	BPH	Chronic prostatitis
	1107/04	68	TURP	BPH	Basalcell hyperplasia-complete, transitional cell metaplas
	1149/04	65	TURP	BPH	Low grade PIN
	1156/04	73	Trucut	Adenocarcinoma Grade II	
	1192/04	60	Trucut	Adenocarcinoma Grade III	High grade PIN
	1193/04	69	TURP	BPH	Leiomyomatous nodule
	1247/04	67	TURP	BPH	Chronic prostatitis
	1248/04	63	TURP	Adenocarcinoma Grade II	Low grade PIN
	1249/04	65	TURP	BPH	Chronic prostatitis
	1274/04	66	TURP	Adenocarcinoma Grade III	Chronic prostatitis
	1288/04	52	TURP	BPH	

ɔ.	Biopsy No.	Age/Male	Biopsy Specimen	Histopathological Diagnosis	Associated Lesions
	1335/04	73	TURP	BPH	
	1418/04	72	Trucut	Adenocarcinoma Grade III	High grade PIN
	1435/04	60	Trucut	Adenocarcinoma Grade II	
	1441/04	65	TURP	BPH	Chronic prostatitis
	1442/04	55	TURP	BPH	Low grade PIN, Chronic prostatitis
	1464/04	72	TURP	BPH	High grade PIN
	1552/04	48	TURP	BPH	Stromal hyperplasia, Leiomyomatous nodule
	1567/04	87	Trucut	BPH	Low grade PIN
	1590/04	65	TURP	BPH	Low grade PIN
	1630/04	80	TURP	BPH	Transitional cell metaplasia
	1631/04	60	TURP	BPH	Chronic prostatitis
	1632/04	80	TURP	BPH	Chronic prostatitis
	1654/04	50	TURP	BPH	Cystic atrophy
	1656/04	53	TURP	BPH	
	1773/04	77	TURP	BPH	Cystic atrophy
	1825/04	70	TURP	BPH	Chronic prostatitis
	1840/04	70	TURP	BPH	Transitional cell metaplasia
	1867/04	64	Trucut	Adenocarcinoma Grade II	
	1871/04	49	TURP	BPH	
	1892/04	72	TURP	BPH	Low grade PIN
	1893/04	65	TURP	BPH	Chronic prostatitis, Abscess
	1996/04	60	TURP	BPH	Corpora amylacea
	2008/04	64	TURP	BPH	Cystic atrophy
	2009/04	73	TURP	BPH	Chronic prostatitis, Atypical adenomatous hyperplasia
	2044/04	52	TURP	BPH	Chronic prostatitis
	2045/04	54	TURP	BPH	
	2168/04	68	TURP	BPH	Low grade PIN
	2206/04	55	Trucut	BPH	Squamous metaplasia
	2272/04	70	Trucut	Inadequate tissue	
	2316/04	78	TURP	BPH	Low grade PIN, chronic prostatitis
	2317/04	50	TURP	BPH	Low grade PIN
	2335/04	71	Trucut	Adenocarcinoma grade III	Chronic prostatitis
	2337/04	70	TURP	BPH	Chronic prostatitis
	2392/04	71	TURP	BPH	Low grade PIN, Clearcell cribriform hyperplasia
	2393/04	75	TURP	BPH	Chronic prostatitis
	2468/04	55	TURP	BPH	
	2470/04	51	TURP	BPH	Low grade PIN, chronic prostatitis
	2499/04	60	TURP	BPH	Stromal nodule, lymphocytic prostatitis
	2506/04	73	TURP	BPH	Chronic prostatitis
	2534/04	54	TURP	BPH	Squamous metaplasia, Chronic prostatitis, Infarction

ɔ.	Biopsy No.	Age/Male	Biopsy Specimen	Histopathological Diagnosis	Associated Lesions
	2537/04	66	TURP	BPH	
	2538/04	75	Open Prostatectomy	BPH	Cystic atrophy, chronic prostatitis
	2542/04	65	Trucut	BPH	
	2570/04	56	TURP	BPH	Chronic prostatitis
	2571/04	66	TURP	BPH	Stromal nodule
	2601/04	80	TURP	BPH	Low grade PIN
	2603/04	50	TURP	BPH	Stromal hyperplasia
	2604/04	52	TURP	BPH	Lymphocytic prostatitis
	2699/04	60	TURP	BPH	Low grade PIN, Basalcell hyperplasia-Incomplete
	2727/04	73	TURP	BPH	Low grade PIN
	2739/04	75	Open Prostatectomy	BPH	Atypical adenomatous hyperplasia
	2740/04	60	TURP	BPH	Cystic atrophy, lyumphocytic prostatitis
	2762/04	60	TURP	BPH	Basalcell hyperplasia-complete, Lowgrade PIN
	2764/04	62	TURP	BPH	Lymphocytic prostatitis
	2805/04	75	Trucut	BPH	
	2870/04	70	TURP	BPH	Transitional cell metaplasia
	2891/04	75	TURP	BPH	Basalcell hyperplasia-Incomplete, Lowgrade PIN
.	2893/04	70	TURP	BPH	Chronic prostatitis
.	2894/04	70	TURP	BPH	
.	2925/04	60	TURP	BPH	High grade PIN
.	2962/04	55	TURP	BPH	Infarction, squamous metaplasia, cystic atrophy
.	2964/04	60	TURP	BPH	Transitional cell metaplasia
.	3001/04	70	TURP	BPH	Stromal nodule, lymphocytic prostatitis
.	3004/04	62	TURP	BPH	
.	3032/04	72	TURP	BPH	Lymphocytic prostatitis
.	3074/04	58	TURP	BPH	Chronic prostatitis
.	3082/04	55	TURP	BPH	Low grade PIN
.	3099/04	64	Trucut	BPH	High grade PIN
.	3107/04	60	TURP	BPH	Stromal nodule, Low grade PIN
.	3162/04	50	TURP	BPH	
.	3182/04	50	Open Prostatectomy	BPH	Cystic atrophy
.	3183/04	69	TURP	BPH	
.	3206/04	60	TURP	BPH	High grade PIN, transitional cell metaplasia
.	3225/04	50	TURP	BPH	High grade PIN
.	3253/04	65	TURP	BPH	Chronic prostatitis
.	3254/04	65	TURP	BPH	
.	3255/04	67	TURP	BPH	Basalcell hyperplasia-complete
.	3268/04	65/M	Trucut	Only fibrocollagenous tissue	
.	3303/04	70	TURP	BPH	High grade PIN
.	3304/04	40	TURP	BPH	High grade PIN

ɔ.	Biopsy No.	Age/Male	Biopsy Specimen	Histopathological Diagnosis	Associated Lesions
.	3305/04	70	TURP	BPH	
.	3327/04	68	TURP	BPH	Low grade PIN, chronic prostatitis
.	3340/04	65	TURP	BPH	Stromal nodule
.	3341/04	70	TURP	BPH	Chronic lymphocytic prostatitis
.	3347/04	69	Trucut	BPH	
.	3370/04	55	TURP	BPH	Corpora amylacea, stromal calcification
.	3380/04	70	TURP	BPH	Chronic prostatitis, cystic atrophy
.	3407/04	60	TURP	BPH	Granulomatous prostatitis
.	3484/04	75	TURP	BPH	
.	3503/04	73	TURP	BPH	Focal basal cell hyperplasia-complete
.	3524/04	65	TURP	BPH	
.	3525/04	65	TURP	BPH	Basal cell hyperplasia-Incomplete
.	3526/04	62	TURP	BPH	
.	3605/04	55	TURP	BPH	Transitional cell metaplasia
.	3616/04	70	TURP	BPH	Atypical adenomatous hyperplasia
.	3617/04	63	TURP	BPH	Low grade PIN
.	3634/04	75	TURP	Adenocarcinoma Grade I	Chronic prostatitis
.	3657/04	69	TURP	BPH	Chronic prostatitis
.	3692/04	73	TURP	BPH	Low grade PIN
.	3693/04	62	TURP	BPH	
.	3694/04	75	TURP	BPH	
.	3773/04	70	TURP	BPH	Stromal hyperplasia
.	3775/04	65	TURP	BPH	Chronic prostatitis
.	3776/04	70	TURP	BPH	Atypical adenomatous hyperplasia
.	3783/04	72	TURP	BPH	Stromal hyperplasia
.	3815/04	75	TURP	BPH	
.	3840/04	60	TURP	BPH	Lymphocytic prostatitis
.	3841/04	60	TURP	BPH	Low grade PIN
.	3931/04	78	TURP	BPH	Low grade PIN
.	4334A/04	55	TURP	BPH	
.	4374/04	70	TURP	BPH	Lymphocytic prostatitis
.	4419/04	64	TURP	BPH	
.	4420/04	70	TURP	BPH	Stromal nodule
.	4421/04	65	TURP	BPH	
.	4479/04	53	Trucut	Adenocarcioma Grade I	Low grade PIN
.	4530/04	72	TURP	Adenocarcioma Grade II	High grade PIN
.	4545/04	75	TURP	BPH	Chronic prostatitis
.	4563/04	62	TURP	BPH	
.	4568/04	70	TURP	BPH	Atypical adenomatous hyperplasia
.	4597/04	50	TURP	BPH	Transitional cell metaplasia

ɔ.	Biopsy No.	Age/Male	Biopsy Specimen	Histopathological Diagnosis	Associated Lesions
.	4629/04	60	TURP	BPH	Chronic prostatitis
.	4630/04	65	Trucut	Adenocarcinoma Grade-II	
.	4631/04	69	TURP	BPH	
.	4633/04	60	TURP	BPH	
.	4708/04	65	Trucut	BPH	Low grade PIN, Postatrophic hyperplasia
.	4717/04	65	TURP	BPH	Basal cell hyperplasia-complete
.	4718/04	64	TURP	BPH	Low grade PIN
.	4834/04	70	Trucut	Only blood clot	
.	4890/04	68	TURP	BPH	Chronic prostatitis
.	4938/04	68	TURP	BPH	Granulomatous prostatitis, Atypical adenomatous hyperpl
.	4941/04	60	TURP	BPH	
.	5020/04	72	TURP	BPH	
.	5023/04	65	TURP	BPH	Low grade PIN
.	5048/04	65	Trucut	Adenocarcinoma Grade-I	High grade PIN
.	5184/04	52	TURP	Adenocarcinoma Grade-II	High grade PIN
.	5185/04	65	TURP	BPH	Chronic prostatitis
.	5206/04	55	TURP	BPH	Atypical adenomatous hyperplasia
.	5208/04	56	TURP	BPH	Basalcell hyperplasia-Incomplete
.	5280/04	65	TURP	BPH	Corpora amylacea
.	5307/04	61	TURP	BPH	
.	5392/04	75	TURP	BPH	Chronic prostatitis, Abscess
.	5461/04	71	TURP	BPH	Transitional cell metaplasia
.	5484/04	53	TURP	BPH	Chronic lymphocytic prostatitis
.	5489/04	70	TURP	BPH	High grade PIN
.	5490/04	72	Trucut	Adenoocarcinoma Grade-I	High grade PIN
.	5518/04	50	TURP	BPH	Chronic prostatitis
.	5519/04	75	TURP	BPH	
.	5520/04	65	Open Prostatectomy	BPH	Cystic atrophy
.	5527/04	67	TURP	BPH	Stromal hyperplasia
.	5549/04	60	TURP	BPH	
.	5550/04	06	TURP	BPH	High grade PIN
.	5632/04	70	TURP	BPH	Chronic prostatitis
.	5650/04	60	TURP	BPH	Atypical adenomatous hyperplasia
.	5657/04	70	Open prostatectomy	BPH	Corpora amylacea
.	5686/04	65	TURP	BPH	Chronic prostatitis, Low grade PIN
.	5712/04	61	TURP	BPH	
.	5713/04	50	TURP	BPH	
.	5760/04	75	TURP	BPH	Chronic prostatitis
.	5780/04	78	TURP	BPH	Chronic prostatitis
.	5786/04	68	TURP	BPH	Low grade PIN

ɔ.	Biopsy No.	Age/Male	Biopsy Specimen	Histopathological Diagnosis	Associated Lesions
.	5809/04	60	TURP	BPH	
.	5829/04	70	TURP	BPH	Atypical adenomatous hyperplasia
.	5830/04	60	TURP	BPH	High grade PIN
.	5854/04	57	Trucut	Adenocarcinoma Grade-II	High grade PIN
.	5861/04	73	TURP	BPH	Chronic prostatits
.	5862/04	60	TURP	BPH	Atypical adenomatous hyperplasia
.	5906/04	68	TURP	BPH	Squamous metaplasia, Infarction
.	5907/04	85	TURP	BPH	
.	5941/04	64	TURP	BPH	Chronic prostatitis
.	5943/04	66	TURP	BPH	Corpora amylacea
.	5978/04	60	TURP	BPH	Chronic prostatitis
.	5979/04	41	TURP	BPH	Acinar atrophy
.	5981/04	82	Open Prostatectomy	BPH	Corpora amylacea, cysticatrophy
.	6056/04	64	TURP	BPH	
.	6057/04	75	TURP	BPH	Stromal nodule, Low Grade PIN
.	6074/04	74	Trucut	Adenocarcinoma Grade-I	Low grade PIN, Chronic prostatitis
.	6076/04	60	TURP	BPH	
.	6077/04	67	TURP	BPH	
.	6078/04	75	Open prostatectomy	BPH	Chronic prostatitis
.	6129/04	71	TURP	BPH	Transitional cell metaplasia, stromal nodule
.	6138/04	77	TURP	BPH	Low grade PIN
.	6153/04	62	TURP	BPH	
.	6220/04	70	Trucut	Only blood clot	
.	6223/04	82	TURP	BPH	Corpora amylacea
.	6225/04	50	Open prostatectomy	BPH	High grade PIN, stromal nodule
.	6226/04	48	TURP	BPH	
.	6273/04	65	TURP	BPH	Chronic prostatitis
.	6301/04	72	TURP	Adenocarcinoma Grade-I	High grade PIN
.	6302/04	70	TURP	BPH	Low grade PIN
.	6367/04	65	TURP	BPH	Chronic prostatitis, Atypical adenomatous hyperplasia
.	6422/04	54	TURP	BPH	
.	6423/04	74	TURP	BPH	Transitional cell metaplasia, lymphocytic prostatitis
.	6487/04	70	TURP	BPH	
.	6491/04	76	TURP	BPH	
.	6492/04	65	TURP	BPH	Stromal nodule
.	6586/04	65	TURP	BPH	Atypical adenomatous hyperplasia
.	6632/04	75	TURP	BPH	Granulomatous prostatitis
.	6635/04	60	TURP	BPH	High grade PIN
.	6675/04	70	TURP	BPH	Chronic prostatitis
.	6676/04	60	TURP	BPH	Low grade PIN

ɔ.	Biopsy No.	Age/Male	Biopsy Specimen	Histopathological Diagnosis	Associated Lesions
.	6694/04	70	Open Prostatectomy	BPH	Cystic atrophy
.	6740/04	53	TURP	BPH	
.	6742/04	67	TURP	BPH	Basalcell hyperplasia-Complete
.	6744/04	70	TURP	BPH	Stromal nodule
.	6767/04	68	TURP	BPH	Chronic prostatitis
.	6768/04	82	TURP	BPH	Chronic prostatitis
.	6861/04	75	TURP	BPH	Bacterial prostatitis, Abscess, High grade PIN
.	6862/04	60	TURP	BPH	Granulomatous prostatitis
.	6903/04	71	TURP	BPH	Squamous metaplasia, Transitionalcell metaplasia, High (
.	6906/04	63	TURP	BPH	
.	6987/04	55	Trucut	Inadequate tissue	
.	6989/04	65	TURP	BPH	Chronic prostatitis
.	6990/04	65	TURP	BPH	Low grade PIN
.	7032/04	70	TURP	BPH	Cystic atrophy
.	7033/04	69	TURP	BPH	Lymphocytic prostatitis
.	7048/04	60	TURP	BPH	Atypical adenomatous hyperplasia
.	7127/04	72	TURP	BPH	
.	7161/04	60	TURP	BPH	Basal cell hyperplasia-Incomplete
.	7162/04	72	TURP	Adenocarcinoma Grade III	High grade PIN, Chronic prostatitis
.	7202/04	62	TURP	BPH	
.	7203/04	70	TURP	BPH	Leiomyomatous nodule
.	7219/04	55	TURP	BPH	Chronic prostatitis
.	7280/04	51	TURP	BPH	
.	7281/04	65	TURP	BPH	Stromal nodule
.	7345/04	70	TURP	BPH	Atypical adenomatous hyperplasia
.	7416/04	65	TURP	BPH	
.	7417/04	71	TURP	BPH	
.	7430/04	75	TURP	BPH	Low grade PIN
.	7431/04	53	TURP	BPH	
.	7451/04	60	TURP	BPH	Chronic prostatitis
.	7452/04	65	TURP	BPH	Infarction, squamous metaplasia, Lymphocytic prostatitis
.	7476/04	67	TURP	BPH	Chronic prostatitis
.	7480/04	50	TURP	BPH	
.	7481/04	71	TURP	BPH	Low grade PIN
.	9485/04	62	TURP	BPH	
.	12/05	76	TURP	BPH	Stromal nodule
.	220/05	67	TURP	BPH	
.	255/05	70	TURP	BPH	Chronic prostatitis, cystic atrophy
.	256/05	70	TURP	BPH	Transitional cell metaplasia
.	257/05	74	TURP	BPH	Chronic prostatitis

ɔ.	Biopsy No.	Age/Male	Biopsy Specimen	Histopathological Diagnosis	Associated Lesions
.	311/05	59	TURP	BPH	High grade PIN
.	313/05	73	TURP	BPH	
.	317/05	63	Trucut	BPH	Atypical adenomatous hyperplasia
.	348/05	70	TURP	BPH	Low grade PIN, Cystic atrophy
.	360/05	60	TURP	BPH	Transitional cell metaplasia
.	361/05	55	Trucut	Adenocarcinoma Grade III	
.	413/05	66	TURP	BPH	Chronic prostatitis
.	415/05	56	TURP	BPH	Chronic prostatitis, Low grade PIN
.	439/05	61	TURP	BPH	
.	440/05	35	TURP	BPH	Basalcell hyperplasia-Incomplete
.	443/05	60	Trucut	BPH	Chronic prostatitis
.	483/05	40	TURP	BPH	Atypical adenomatous hyperplasia
.	485/05	51	TURP	BPH	
.	547/05	55	TURP	BPH	Chronic lymphocytic prostatitis
.	588/05	77	Trucut	Only haemorrhage	
.	589/05	60	Trucut	Only bladder mucosa	
.	594/05	55	TURP	BPH	Low grade PIN
.	595/05	60	TURP	BPH	Chronic prostatitis
.	623/05	80	TURP	BPH	Basalcell hyperplasia-complete
.	624/05	72	TURP	BPH	Squamous metaplasia, Infarction
.	666/05	58	TURP	BPH	Low grade PIN
.	671/05	60	TURP	BPH	Chronic prostatitis
.	681/05	75	Trucut	Inadequate tissue	
.	722/05	70	TURP	BPH	High grade PIN
.	723/05	52	TURP	BPH	Acinar atrophy, stromal nodule
.	727/05	70	TURP	BPH	
.	787/05	47	Trucut	BPH	Chronic prostatitis
.	788/05	55	TURP	BPH	
.	815/05	71	TURP	BPH	Low grade PIN
.	858/05	65	Trucut	Adenocarcinoma Grade-II	High grade PIN, Lymphocytic prostatitis
.	859/05	75	Trucut	Few prostatic glands	
.	866/05	66	TURP	BPH	Cystic atrophy
.	927/05	77	Trucut	Adenocarcinoma Grade-I	Chronic prostatitis
.	959/05	70	TURP	BPH	Basalcell hyperplasia-Incomplete
.	1011/05	75	TURP	BPH	Chronic prostatitis
.	1013/05	61	TURP	BPH	Low grade PIN
.	1036/05	56	TURP	BPH	
.	1037/05	65	TURP	BPH	Corpora amylacea
.	1067/95	76	TURP	BPH	Tuberculous granulomatous prostatitis
.	1127/05	42	Trucut	BPH	

ɔ.	Biopsy No.	Age/Male	Biopsy Specimen	Histopathological Diagnosis	Associated Lesions
.	1129/05	71	TURP	BPH	Low grade PIN
.	1153/05	70	TURP	BPH	Transitional cell metaplasia, squamous metaplasia
.	1193/05	70	TURP	BPH	Basal cell hyperplasia-complete
.	1194/05	65	TURP	BPH	
.	1304/05	77	TURP	BPH	Cystic atrophy
.	1305/05	60	TURP	BPH	Chronic prostatitis
.	1337/05	75	TURP	BPH	High grade PIN
.	1338/05	55	TURP	Adenocarcinoma Grade-II	Low grade PIN
.	1369/05	65	TURP	BPH	
.	1370/05	60	TURP	BPH	Stromal hyperplasia
.	1430/05	70	TURP	BPH	
.	1486/05	84	TURP	BPH	Low grade PIN
.	1490B/05	68	TURP	BPH	
.	1602A/05	70	TURP	BPH	
.	1603/05	60	TURP	Adenocarcinoma Grade-IV	Chronic prostatitis, High grade PIN
.	1672/05	50	TURP	BPH	Low grade PIN
.	1675/05	75	TURP	BPH	Chronic prostatitis
.	1696/05	62	Open Prostatectomy	BPH	Cystic atrophy
.	1697/05	59	TURP	BPH	Basalcell hyperplasia-Incomplete
.	1698/05	62	TURP	BPH	
.	1743/05	63	TURP	BPH	Chronic prostatitis
.	1814/05	70	TURP	BPH	Basalcell hyperplasia-complete
.	1818/05	55	TURP	BPH	
.	1859/05	76	Trucut	BPH	Atypical adenomatous hyperplasia
.	1886/05	68	TURP	BPH	
.	1887/05	80	TURP	BPH	Basalcell hyperplasia-Incomplete
.	1922/05	52	Trucut	BPH	Prostatitis, Low grade PIN
.	1946/05	67	TURP	BPH	
.	1948/05	61	TURP	BPH	Chronic prostatitis
.	1949/05	60	TURP	BPH	Chronic prostatitis
.	1993/05	70	Turcut	Adenocarcinoma Grade-II	High grade PIN
.	2037/05	65	TURP	BPH	Cystic atrophy
.	2068/05	72	Trucut	BPH	
.	2083/05	60	TURP	BPH	Transitional cell metaplaisa
.	2100/05	74	TURP	BPH	
.	2156/05	80	TURP	BPH	Stromal hyperplasia
.	2176/05	55	TURP	BPH	
.	2204/05	51	TURP	BPH	Atypical adenomatous hyperplasia
.	2263/05	65	TURP	BPH	Low grade PIN
.	2276/05	60	TURP	BPH	Chronic prostatitis

ɔ.	Biopsy No.	Age/Male	Biopsy Specimen	Histopathological Diagnosis	Associated Lesions
.	2334/05	60	TURP	BPH	Cystic atrophy
.	2335/05	70	TURP	BPH	Chronic prostatitis
.	2356/05	54	TURP	BPH	Fungal prostatitis
.	2357/05	70	TURP	BPH	
.	2406/05	55	TURP	BPH	Stromal hyperplasia
.	2492/05	60	TURP	BPH	Transitional cell metaplasia
.	2493/05	67	TURP	BPH	Low grade PIN
.	2495/05	61	TURP	BPH	Chronic prostatitis
.	2517/05	65	TURP	BPH	Basalcell hyperplasia-complete
.	2518/05	63	TURP	BPH	Chronic prostatitis
.	2574/05	75	TURP	BPH	
.	2634/05	74	Trucut	BPH	Low grade PIN, postatrophic hyperplasia
.	2636/05	45	Trucut	Only fibrous tissue	
.	2637/05	43	TURP	BPH	
.	2638/05	69	TURP	BPH	Squamous metaplasia, Infarction
.	2718/05	75	TURP	BPH	Low grade PIN
.	2740/05	75	TURP	BPH	Chronic prostatitis
.	2742/05	73	TURP	BPH	
.	2783/05	53	TURP	BPH	Chronic prostatitis
.	2807/05	55	TURP	BPH	
.	2861/05	75	TURP	BPH	High grade PIN
.	2881/05	75	TURP	BPH	
.	2882/05	77	TURP	BPH	Low grade PIN
.	2920/05	75	Trucut	BPH	Low grade PIN
.	2925/05	55	TURP	BPH	Chronic prostatitis, Basalcell hyperplasia-Incomplete
.	2926/05	66	TURP	BPH	Chronic prostatitis
.	2942/05	75	TURP	BPH	
.	2945/05	60	TURP	BPH	Cystic atrophy
.	2947/05	32	TURP	BPH	
.	3018/05	50	TURP	Adenocarcinoma grade II	Chronic prostatitis
.	3058/05	62	TURP	BPH	Stromal nodule, Chronic prostatitis
.	3125/05	65	TURP	BPH	Squamous metaplasia
.	3128/05	80	TURP	BPH	Stromal hyperplasia
.	3139/05	65	Trucut	Only few prostatic glands	
.	3207/05	65	TURP	BPH	
.	3234/05	72	TURP	BPH	Chronic prostatitis
.	3351/05	65	TURP	BPH	
.	3353/05	55	TURP	BPH	Basalcell hyperplasia-complete
.	3385/05	75	TURP	BPH	Cystic atrophy
.	3386/05	60	TURP	BPH	Chronic prostatitis

ɔ.	Biopsy No.	Age/Male	Biopsy Specimen	Histopathological Diagnosis	Associated Lesions
.	3411/05	72	TURP	BPH	Transitional cell metaplasia
.	3413/05	51	TURP	BPH	
.	3414/05	60	TURP	BPH	
.	3470/05	72	TURP	BPH	Corpora amylacea
.	3484/05	55	TURP	BPH	Chronic prostatitis
.	3485/05	75	TURP	BPH	Corpora amylacea
.	3563/05	67	TURP	BPH	High grade PIN, corpora amylacea
.	3591/05	54	Trucut	BPH	Atypical adenomatous hyperplasia
.	3616/05	65	TURP	BPH	Basalcell hyperplasia-complete
.	3657/05	64	TURP	BPH	Chronic prostatitis
.	3681/05	65	TURP	BPH	Low grade PIN
.	3702/05	70	Open Prostatectomy	BPH	Atypical adenomatous hyperplasia
.	3704/05	60	TURP	BPH	
.	3705/05	60	TURP	BPH	Chronic prostatitis
.	3746/05	64	Trucut	Adenocarcinoma Grade III	High grade PIN
.	3755/05	75	Trucut	Adenocarcinoma Grade III	High grade PIN
.	3757/05	85	TURP	BPH	Cystic atrophy
.	3783/05	65	TURP	BPH	
.	3784/05	55	TURP	BPH	Transitional cell metaplasia
.	3857/05	70	TURP	Adenocarcinoma grade II	Chronic prostatitis, Low grade PIN
.	3858/05	70	TURP	BPH	Chronic lymphocytic prostatitis
.	3859/05	76	TURP	BPH	Corpora amylacea
.	3913/05	40	TURP	BPH	Stromal hyperplasia
.	3929/05	60	Trucut	Adenocarcinoma grade V	Chronic prostatitis
.	3935/05	80	TURP	BPH	Low grade PIN
.	3936/05	72	TURP	Adenocarcinoma Grade I	Chronic prostatitis
.	3979/05	70	TURP	BPH	
.	4011/05	72	TURP	BPH	Chronic prostatitis
.	4012/05	86	TURP	BPH	
.	4013/05	57	TURP	BPH	Stromal nodule
.	4067/05	63	TURP	BPH	Chronic prostatitis
.	4092/05	73	Trucut	Only few prostatic glands	
.	4095/05	69	TURP	BPH	Low grade PIN
.	4130/05	65	TURP	BPH	High grade PIN
.	4163/05	52	TURP	BPH	
.	4164/05	63	TURP	BPH	Chronic prostatitis
.	4227/05	75	TURP	BPH	
.	4239/05	70	TURP	BPH	Cystic atrophy
.	4263/05	65	Trucut	Adenocarcinoma Grade III	Chronic prostatitis, Low grade PIN
.	4283/05	65	TURP	BPH	Chronic prostatitis

ɔ.	Biopsy No.	Age/Male	Biopsy Specimen	Histopathological Diagnosis	Associated Lesions
.	4335/05	65	TURP	BPH	
.	4361/05	54	TURP	BPH	Xantho granulomatous prostatitis
.	4362/05	50	TURP	BPH	Corpora amylacea
.	4488/05	68	TURP	BPH	Chronic prostatitis
.	4543/05	50	TURP	BPH	Chronic prostatitis
.	4700/05	60	TURP	BPH	Low grade PIN
.	4726/05	67	TURP	BPH	Basalcell hyperplasia-Incomplete
.	4728/05	45	TURP	BPH	
.	4730/05	55	TURP	BPH	Stromal hyperplasia
.	4752/05	73	TURP	BPH	Atypical adenomatous hyperplasia
.	4753/05	57	TURP	BPH	Granulomatous prostatitis, Clearcell cribriform hyperplasi
.	4770/05	75	TURP	BPH	High grade PIN
.	4847/05	82	TURP	BPH	Cystic atrophy
.	4897/05	57	TURP	BPH	
.	4898/05	65	TURP	BPH	
.	4899/05	85	TURP	BPH	Chronic prostatitis, Low grade PIN
.	4900/05	65	TURP	BPH	
.	4912/05	72	Trucut	Adenocarcinoma Grade III	High grade PIN
.	4986/05	75	TURP	BPH	Stromal nodule
.	4987/05	70	TURP	BPH	Chronic prostatitis
.	5043/05	64	TURP	BPH	Transitional cell metaplasia
.	5044/05	60	TURP	BPH	Corpora amylacea
.	5071/05	60	TURP	BPH	
.	5073/05	72	TURP	BPH	
.	5113/05	66	TURP	Adenocarcinoma Grade IV	Chronic prostatitis, High grade PIN
.	5114/05	54	TURP	BPH	Chronic prostatitis
.	5138/05	52	TURP	BPH	High grade PIN
.	5139/05	65	TURP	BPH	
.	5179/05	65	TURP	BPH	Low grade PIN
.	5198/05	80	TURP	BPH	Chronic prostatitis
.	5210/05	72	TURP	BPH	
.	5224/05	86	TURP	Adenocarcinoma grade I	Low grade PIN
.	5248/05	65	TURP	BPH	Chronic prostatitis
.	5278/05	78	TURP	BPH	Stromal hyperplasia
.	5337/05	60	TURP	BPH	Basalcell hyperplasia-complete, Chronic prostatitis
.	5339/05	57	TURP	BPH	Chronic prostatitis
.	5342/05	55	TURP	Adenocarcinoma Grade III	High grade PIN
.	5366/05	50	TURP	BPH	Chronic prostatitis
.	5368/05	53	TURP	BPH	
.	5373/05	64	TURP	BPH	Low grade PIN

ɔ.	Biopsy No.	Age/Male	Biopsy Specimen	Histopathological Diagnosis	Associated Lesions
.	5376/05	54	TURP	BPH	Squamous metaplasia
.	5453/05	67	TURP	BPH	Low grade PIN
.	5480/05	61	TURP	BPH	Chronic prostatitis
.	5551/05	65	TURP	BPH	
.	5552/05	45	TURP	BPH	
.	5556/05	70	TURP	BPH	High grade PIN
.	5580/05	65	TURP	BPH	Cystic atrophy
.	5602/05	56	TURP	BPH	High grade PIN
.	5660/05	60	TURP	BPH	Chronic prostatitis
.	5663/05	75	TURP	BPH	Chronic lymphocytic prostatitis, Abscess
.	5664/05	60	TURP	BPH	Low grade PIN
.	5706/05	70	TURP	BPH	Chronic prostatitis
.	5766/05	75	Trucut	Adenocarcinoma grade IV	
.	5792/05	50	TURP	BPH	
.	5793/05	65	TURP	BPH	
.	5799/05	65	TURP	BPH	
.	5818/05	50	Trucut	Only fibromuscular tissue	
.	5840/05	70	TURP	Only few prostatic glands	
.	5880/05	80	TURP	BPH	
.	5881/05	63	TURP	BPH	Chronic prostatitis
.	5907/05	63	TURP	BPH	Chronic prostatitis
.	5909A/05	82	TURP	BPH	
.	5968/05	75	Trucut	BPH	Low grade PIN
.	6036/05	70	TURP	BPH	
.	6084/05	50	Trucut	Tiny tissue - few glands	
.	6085/05	65	Trucut	Tiny tissue - few glands	
.	6090/05	65	TURP	BPH	
.	6269A/05	72	TURP	BPH	Cystic atrophy
.	6301/05	82	TURP	BPH	Transitional cell metaplasia
.	6323/05	45	TURP	BPH	
.	6324/05	56	TURP	BPH	High grade PIN
.	6326/05	65	Trucut	BPH	Chronic lymphocytic prostatitis
.	6374/05	72	TURP	BPH	Basalcell hyperplasia-Incomplete
.	6380/05	81	TURP	BPH	
.	6409/05	66	TURP	BPH	Cystic atrophy
.	6412/05	68	TURP	BPH	Stromal nodule
.	6441/05	72	TURP	BPH	Low grade PIN
.	6466/05	64	TURP	BPH	Chronic prostatitis

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